the salary of our less privileged Black colleagues (Africans) to our level. With the assistance of Black doctors, mainly G.P.s, in the Transvaal Study Circle we started the Salary Equalization Fund. To publicize this fund we wrote a letter to the *South African Medical Journal*. An altered version was published. "African" was replaced with "Bantu" in all sentences. Response to this letter from White doctors was negligible. When we made our first payments to the Africans in December 1972 less than 20% of the contributions had come from Whites.—I am, etc.,

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Making Hospital Geriatrics Work

SIR,—Dr. R. W. Parnell (24 November, p. 484) suggests that there will be a breakdown in the hospital emergency services if a rapid increase in the provision of geriatric services is not achieved. This would seem to be supported by Dr. R. V. Boyd's figures for Greenwich (3 November, p. 298).

Alternative methods of bed usage in both the hospital and community residential services may be an answer in regions where a rapid increase in the total bed complement to the national norm seems unlikely. The use of "linked" beds may overcome some of the problems which lead to the admission of geriatric patients into medium- or long-stay beds. The use of the "relief" admission bed for short periods is already established in our hosiptal and local authority units.

It may be possible to extend this concept to other forms of relief bed. This could comprise the use of beds for a day or a night, for three or four days, for one week in and one week out, etc. This is seen as an extension of the present rapid increase in day care provisions, especially where the patient is too disabled or demanding for the latter to be effective. Staffing and transport structures would need to be modified to cope with the increased work load, but morale of both staff and patients is less likely to "plummet" than in dealing with a predominantly long-stay population. We may also move nearer the ideal of maintaining the majority of the elderly sick in the home.—I am, etc.,

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Home Graduates Only

SIR,—Of late it has become increasingly common for doctors in Britain to prefer graduates of British universities only to fill vacancies in their group practices. In a recent issue of the *B.M.J.*, out of 33 advertisements, only five did not specify this condition. One fails to understand the rationale behind their thinking. What is it they find so undesirable in overseas-trained medical graduates? These foreign graduates are good enough to work in N.H.S. hospitals in various specialties, many of them in responsible positions, and may have spent years in various branches of medicine. A minority of these doctors may decide to enter general practice, but they find the doors shut in their face. Why is it so? Is this because the small island of Britain is overdoctored? Or are these doctors not good enough to treat patients outside hospital? If this is the case, then overseas graduates need no longer be entertained for N.H.S. posts in hospitals.

The present generation of Britons need no longer suffer from any moral obligation towards its ex-colonial peoples. Many of these ex-colonial countries have not had enough time to organize matters. They are still afflicted with the mistakes of their past rulers and the problems facing the present rulers. It will be many more years before their houses can be set in order. After all, how many years did it take Britain to achieve its present standard of a welfare state?

One must realize that it is a period of transition for many of these overseas countries. If their people decide to settle abroad, it is for the same reason that many Britons emigrate abroad—for example, lack of opportunities at home, a desire to improve their standard of living, and a sense of adventure. This trend has been going on for many years and will continue to do so until and unless every one of us begins to feel a sense of patriotism and nationalism and decides to remain at home, for better or worse. If that should ever happen, then the whole concept of international understanding and cooperation is a farce, to say the least.—I am, etc.,

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High Dose Frusemide in Established Acute Renal Failure

SIR,—I was interested to read the paper by Professor F. Cantarovich and others (24 November, p. 449). This work is commendable and their regimen is similar to the one which I have adopted in the management of established renal failure. In the management of acute oliguric renal failure I found that in four out of 12 patients diuresis occurred and dialysis was averted. In the remaining eight patients frusemide failed to produce a diuresis and dialysis was used.[1]

However, the authors' statement that the increase in the severity of renal failure admitted to dialysis centres could be explained not only by the prophylactic action of frusemide (in patients with reversible oliguria capable of developing an acute renal failure) but also through a therapeutic effect of the drug exerted in cases of less severe acute renal failure which do not require dialytic procedures owing to a shortening of the anuric period is subject to question. It has been my procedure to give frusemide intravenously slowly over a period of 10 minutes, initially in a dosage of 250 mg/l. If successful, diuresis usually begins in 3-4 minutes, and at least 100 ml is generally passed in the first hour. If diuresis does not occur within the first hour preparations for dialysis are begun. Previously no further frusemide was given, but of late I have continued frusemide in a dose of 1-2 g daily even though dialysis has been initiated. The severity of acute renal failure seems to be related to the cause of the acute renal failure; in our experience it is usually due to septic abortion (with liver necrosis, septicaemia, and acute tubular necrosis) or the ingestion of herbal medicines which cause both liver and kidney damage. These factors obviously affect the mortality. In another series sepsis, age, and surgical operations have been incriminated as affecting mortality.[2]

Finally, in our experience with frusemide in acute renal failure it is not uncommon to observe dependence of patients on frusemide. Thus though diuresis may occur on giving frusemide in large doses, once frusemide is stopped the urine output diminishes to under 400 ml/24 hr. It may be necessary to continue frusemide in large doses for a period of 3-4 months until recovery of renal function (glomerular filtration rate above 20 ml/min.) is obtained. A detailed report of our experience on acute renal failure is in preparation.—I am, etc.,

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1 Seedat, Y. K., *South African Medical Journal*, 1972, **46**, 1371.
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F.D.P. Levels in Intravascular Haemolysis

SIR,—I am grateful to Dr. P. M. Mannucci (24 November, p. 492) for clarifying the question of intravascular coagulation in haemolysis by reference to favism, in which situation he confirms that disseminated intravascular coagulation (D.I.C.) does not occur but apparently fibrinolysis is depressed.

At the same time he has raised further points which require comment. Firstly, I did not say that defective fibrinolysis precipitates D.I.C. Defective fibrinolyisis in the presence of D.I.C. means that histological evidence of fibrin thrombi and tissue damage are likely to be found.[1]

The more important question is why does D.I.C. occur only in certain haemolytic situations, such as transfusion of incompatible blood, in drowning, and after ingestion of strong acids? The answers are that in incompatible blood transfusion D.I.C. is initiated by the immune reaction,[2] that in drowning anoxia and acidosis cause the D.I.C., though water haemolysis does not,[3] and that corrosive acids cause D.I.C. directly[4] and by damaging vascular endothelium, but again it is not the haemolysis that matters.—I am, etc.,

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1 Wardle, E. N., *Thrombosis et Diathesis Haemorrhagica*, 1973, **29**, 579.
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3 Slaastad, R. A., and Eika, C., *Scandinavian Journal of Haematology*, 1973, **11**, 217
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Analgesics and the Kidney

SIR,—We feel that the views put forward by Dr. I. C. Calder and his colleagues (22 September, p. 633) are not in accord with the observable pathology of analgesic damage to the kidney in the human. It has been shown by a number of observers[1-3] that the essential process is a progressive damage to the papilla commencing near the tip and involving the elements closest to the collecting ducts, possibly the ascending loops of Henle first. For a long time the collecting ducts survive, and it is usually only at the stage when part of the papilla is totally